Abstract

The effects of long- term treatment with hydrogen sulfide on memory retention of inhibitory avoidance learning in the 6-hydroxydopamine induced Parkinson's

Parkinson's disease (PD) is a neurodegenerative disorder that particularly impairs motor and cognitive function. Hydrogen sulfide, a novel neuromodulator, has neuroprotective effects and regulates learning and memory. It has be shown that Hydrogen sulfide ameliorates homocysteine-induced impairment in cognitive function. The aim of this study was to investigate the potential neuroprotective effects of sodium hydrosulfide (NaHS), as an H_2S donor, on memory retention of inhibitory avoidance learning in an unilateral 6-hydroxydopamine (6-OHDA) rat model of PD.

Methods: Male Wistar rats were subjected to unilateral injection of 6-OHDA (30 μg) into the medial forebrain bundle (MFB) and treated with NaHS for 25 days. Animals were divided: control, sham, 6-OHDA, 6-OHDA plus vehicle (saline) and 6-OHDA plus NaHS (2.8 and 5.6 mg/kg, ip). All rats were trained in shuttle box .Electric shocks in the dark chamber were provided via grid floor (50 Hz, 3 s, and 1 mA intensity) by a standard stimulator.

Results: The step-through latency in the Passive avoidance performance test in the control group were significantly higher than those measured in the 6-OHDA group ($P<0.05$), whereas such significant differences were not found between the control and 6-OHDA plus NaHS groups. The results also showed that pre-training injection of NaHS increased the total time spent in the light chamber and decreased the total time spent in the dark chamber in STZ+ NaHS treated rats.

Conclusion: Treatment with NaHS (2.8 and 5.6 mg/kg) increased the STL in the unilateral 6-OHDA model of PD rats, which indicated that H_2S reverses the impairment of learning and memory induced by unilateral injection of 6-OHDA into the MFB.There for NaHS is useful for treatment of memory impairment in PD.

Keywords: Hydrogen sulfide; 6-hydroxydopamine; passive avoidance learning; Medial forebrain bundle.